

## COMMENTARY

# Effective dose: a flawed concept that could and should be replaced

D J BRENNER, PhD, DSc

Center for Radiological Research, Columbia University Medical Center, New York, NY 10032, USA

**ABSTRACT.** The effective dose is designed to provide a single number proportional to the radiobiological “detriment” from a particular, often inhomogeneous, radiation exposure, with detriment representing a balance between carcinogenesis, life shortening and hereditary effects. It is commonly used to allow a comparison of the risks associated with different spatial dose distributions produced by different imaging techniques. The effective dose represents questionable science: two of the most important reasons for this are that the tissue-specific weighting factors used to calculate effective dose are a subjective mix of different endpoints, and that the marked and differing age dependencies for different endpoints are not taken into account. Importantly, the effective dose is prone to misuse, with widespread confusion between effective dose, equivalent dose and absorbed dose. It is suggested here that effective dose could and should be replaced by a new quantity that does not have these problems. An appropriate new quantity could be “effective risk”, which, like effective dose, is a weighted sum of equivalent doses to different tissues; unlike effective dose, where the tissue-dependent weighting factors are a set of subjective committee-defined numbers, the weighting factors for effective risk would simply be evaluated tissue-specific lifetime cancer risks per unit equivalent dose. The resulting quantity would perform the same comparative role as effective dose; it would have the potential to be age- and, if desired, gender-specific, just as easy to estimate, less prone to misuse, more directly interpretable, and based on more defensible science.

Received 12 November 2007  
Revised 16 January 2008  
Accepted 21 February 2008

DOI: 10.1259/bjr/22942198

© 2008 The British Institute of Radiology

## The problem

The effective dose concept [1] represents an attempt to provide a single number that is proportional to the radiobiological “detriment” from a particular radiation exposure, with detriment representing a balance between carcinogenesis, life shortening and hereditary effects. Specifically, it is the sum of the equivalent doses to a number of radiosensitive organs/tissues, with each organ/tissue being weighted by a committee-determined tissue weighting factor. The effective dose is commonly used in radiology to allow comparisons of the risks associated with different spatial dose distributions produced by different imaging techniques.

However, as recently discussed in this journal and elsewhere by Martin [2, 3], the use of the effective dose concept inherently involves a number of problematic assumptions and issues. Perhaps the three most important are:

1. The tissue weighting factors represent a committee-determined subjective balance between the different stochastic endpoints of cancer incidence, cancer mortality, life shortening and hereditary risk. These

weighting factors change every decade or so [1, 4, 5]. As an example, the weighting factor for the gonads has dropped from 0.25 in 1977 [1] to 0.08 in 2007 [5]; in a second example, the carcinogenesis endpoint was represented by cancer mortality in the 1990 weighting factors [4], but by cancer incidence in the 2007 weighting factors [5]. The reasons for such changes are generally less an improvement in our knowledge of radiation risks, and more that different groups of experts will naturally have somewhat differing views on the relative importance of the different endpoints that comprise the radiation-induced “detriment” [6].

2. A second major problem with the effective dose concept is that it is defined to be independent of age at exposure, whereas data suggest that attributable radiation risks are often highly age-dependent, and that risks for different endpoints have different age-at-exposure dependencies [7]. Table 1 provides some examples of paediatric, adult and all-ages site-specific lifetime cancer risks estimates (averaged over the appropriate population age distributions, based on data in the recent Biological Effects of Ionizing Radiation (BEIR)-VII report [8]) and shows very different age-at-exposure dependencies for different cancers. For example, the ratio of paediatric to adult risks for breast cancer is very different from the

Address correspondence to: D J Brenner, Center for Radiological Research, Columbia University Medical Center, New York NY 10032, USA. E-mail: djb3@columbia.edu

**Table 1.** Examples of site-specific, gender-averaged, radiation-attributable lifetime cancer incidence risks per unit equivalent dose, in a Western population, based on Table 12D-1 of the 2006 BEIR-VII report [8]. The BEIR-VII report-predicted age-at-exposure dependencies have been averaged over the 2000 USA census age distributions for individuals exposed as children (under 16 years), adults (over 15 years), and for all ages. The risks are shown per 100 mSv per 100 000 individuals.

Tissue	Children	Adults	All ages
Stomach <sup>a</sup>	66	30	37
Lung <sup>a</sup>	373	166	208
Colon <sup>a</sup>	203	96	118
Liver <sup>a</sup>	32	14	18
Bladder <sup>a</sup>	153	75	91
Uterus <sup>a,b</sup>	37	14	19
Ovary <sup>a,b</sup>	76	28	37
Prostate <sup>a,b</sup>	67	34	41
Breast <sup>b</sup>	865	160	299
Thyroid	200	18	54
Leukemia	133	68	82

<sup>a</sup>It may be noted that the BEIR-VII report [8] used exactly the same age-at-exposure dependency for each of these cancer sites. If, for example, site-specific age-at-exposure dependencies (based on current A-bomb data [7]) were used, the estimated attributable risks would be almost the same for adults vs children for lung cancer, but would still be very different for breast cancer.

<sup>b</sup>Not gender averaged.

corresponding ratio for lung cancer. The implication here is that, for example, two different imaging techniques, which result in equal radiation risks for adults, would generally not result in equal risks for children.

3. A third major problem with the effective dose concept is a practical one, in that it is often confused and misused. Both equivalent dose, which refers to a given tissue, and effective dose, which is a weighted average over the entire body, are measured in Sieverts. So it is no surprise, for example, that the literature on CT contains many examples where the effective dose, the equivalent dose and even the absorbed dose have been confused with one another. This is not a minor matter of semantics — for a typical CT scan, the effective dose is typically about one-third of the maximum equivalent dose.

## A potential solution

Martin [2], while pointing out these and other problems with the effective dose concept, concluded that “there are uncertainties in the quantity E [effective dose], but it is the only quantity available that provides a dose related to the risk of health detriment”. Rather than just live with this flawed concept, it is argued here that we should define a new, simple, less confusing, easy-to-estimate quantity, based on defensible science, which more directly does the job of comparing the risks associated with different inhomogeneous doses. In

contrast to effective dose, such a new quantity should ideally have the following properties:

1. The new quantity should only refer to cancer risks. There is not a logical way by which cancer risks and hereditary risks can be combined into a single number. Although inclusion of hereditary risks was reasonable in the context of what was known in the 1970s [1], our current understanding is that radiation-induced hereditary risks are much smaller than the corresponding cancer risks [5]. Coupled with the essential impossibility of combining cancer and hereditary risks into a single number, it seems appropriate to drop hereditary risks altogether, and focus only on radiation-induced cancer risks. In this context, the cancer-risk endpoint should ideally not be an arbitrary average of cancer incidence, cancer mortality and years of life lost; because the A-bomb cancer incidence data generally contain less potential for bias compared with mortality data [9], it would be reasonable to choose cancer incidence.
2. The new quantity should be age dependent, or at least distinguish between paediatric and adult risks. Arguably, the quantity should also be gender dependent. Gender does not have as big an effect as age, but women are (overall) significantly more sensitive than men, and the potential illogicalities of, for example, including the female breast risk for a male population would be avoided.
3. The new quantity should be easily interpretable, so that it is less prone to misapplication.

These are properties that one would ideally want of a quantity describing low-dose radiation risks from an inhomogeneous dose distribution, which would allow science-based comparisons between different exposure scenarios. It is the purpose of this Commentary to suggest that, rather than using some surrogate for risk, which is effectively what the effective dose is designed to be, it would be perfectly straightforward to use risk itself — such a quantity would meet all of our desired criteria.

Specifically, the currently used effective dose is defined as

$$E = \sum_T w_T H_T, \quad (1)$$

where  $H_T$  are the tissue-specific equivalent doses in tissue T, and  $w_T$  are the committee-defined dimensionless tissue-specific weighting factors. To define a new quantity with the properties described above, we need simply replace the committee-generated tissue weighting factor,  $w_T$ , with organ-specific radiation-induced cancer risk. The resulting quantity — we could call it “effective risk” — would be defined as:

$$R = \sum_T r_T H_T, \quad (2)$$

\*Note that the issues relating to the appropriate dose range in which the “effective risk” concept should be applied are exactly the same as those relating to effective dose, and are beyond the scope of this commentary.

where  $r_T$  are lifetime radiation-attributable tissue-specific cancer risks (per unit equivalent dose to tissue T), examples of which are shown in Table 1. The effective risk,  $R$ , would then be a lifetime radiation-attributable cancer risk.

In other words, instead of multiplying organ doses by committee-devised numbers, let us instead simply multiply organ doses by the best-available, organ-specific lifetime cancer risks. By comparing the two equations, it is clear that this "effective risk",  $R$ , would be no harder to calculate than the effective dose,  $E$ , and, as argued here, it would be scientifically more defensible\*.

As discussed above, it would make sense to define the effective risk quantity,  $R$ , to be dependent on the age-at-exposure range. This would be easy to do; as appropriate, one could use paediatric risks, adult risks or all-ages risks (see Table 1), depending on the application. Although not explicitly stratified in Table 1, it would be quite feasible to make the effective risk gender specific, if desired.

Apart from being scientifically more defensible than the effective dose, an effective risk would be easier to interpret: it would be an effective lifetime radiation-attributable cancer risk ("× per 100 000" individuals, for example), and so would be much more intuitively interpretable than a quantity in Sieverts. This direct interpretability, in terms of risk, might well represent considerable added value [10] in the context of efforts among the medical community [11, 12] to limit and optimize medically based radiation exposures.

The bottom line is that there is a need for a quantity that simply compares the risks from different inhomogeneous dose distributions. However, the effective dose is confusing and is based on flawed science. Let us consider replacing it with a quantity that is just as easy to estimate — an effective risk — which does the same job, is less prone to misuse, is more directly understandable, and is based on more defensible science.

## References

1. International Commission on Radiological Protection. Recommendations of the International Commission on Radiological Protection: ICRP Publication 26. Oxford, UK: Pergamon Press; 1977.
2. Martin CJ. Effective dose: how should it be applied to medical exposures? *Br J Radiol* 2007;80:639–47.
3. Martin CJ. The application of effective dose to medical exposures. *Radiat Prot Dosimetry* 2008, in press.
4. International Commission on Radiological Protection. 1990 Recommendations of the International Commission on Radiological Protection: ICRP Publication 60. Oxford, UK: Pergamon Press; 1991.
5. International Commission on Radiological Protection. Recommendations of the ICRP: ICRP Publication 103. *Annals of the ICRP* 2007; 37.
6. Streffer C. The ICRP 2007 recommendations. *Radiat Prot Dosimetry* 2008, in press.
7. Preston DL, Ron E, Tokuoka S, Funamoto S, Nishi N, Soda M, et al. Solid cancer incidence in Atomic Bomb survivors: 1958–1998. *Radiat Res* 2007;168:1–64.
8. NRC. Health Risks from Exposure to Low Levels of Ionizing Radiation — BEIR VII. Washington, DC: The National Academies Press; 2006.
9. Brenner DJ, Doll R, Goodhead DT, Hall EJ, Land CE, Little JB, et al. Cancer risks attributable to low doses of ionizing radiation: assessing what we really know. *Proc Natl Acad Sci USA* 2003;100:13761–6.
10. Lee CI, Haims AH, Monico EP, Brink JA, Forman HP. Diagnostic CT scans: assessment of patient, physician, and radiologist awareness of radiation dose and possible risks. *Radiology* 2004;231:393–8.
11. Amis ES Jr, Butler PF, Applegate KE, Birnbaum SB, Brateman LF, Hevezi JM, et al. American College of Radiology white paper on radiation dose in medicine. *J Am Coll Radiol* 2007;4:272–84.
12. Brenner DJ, Hall EJ. Computed tomography — an increasing source of radiation exposure. *N Engl J Med* 2007;357:2277–84.